4.1

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION 10
1200 Sixth Avenue
Seattle, Washington 98101

Reply To
Attn Of: ECL-111

May 15, 1997

Reply to
Attn. of HW-113

Charles Preston
Environmental Specialist
Kaiser Aluminum and Chemical Corporation
East 2107 Hawthorne Road
Mead, Washington 99021

Re: Transmittal of Risk Assessment

Dear Mr. Preston:

I have attached the Streamlined Human Health Risk Assessment prepared for EPA by E&E. The document shows that the Removal Action at the site has left the site at acceptable risk levels. This document should be attached to the Completion Report as an Appendix.

If you have any questions or comments please do not hesitate to call me at (206) 553-2106.

Sincerely,

Kevin Rochlin
Project Manager

Attachment

cc: Alex Tula

4.1

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION 10
1200 Sixth Avenue
Seattle, Washington 98101

STREAMLINED HUMAN HEALTH RISK ASSESSMENT SPOKANE JUNKYARD AND ASSOCIATED PROPERTIES SPOKANE, WASHINGTON

Prepared by Ecology and Environment, Inc. for the Environmental Protection Agency
May 1997

STREAMLINED HUMAN HEALTH RISK ASSESSMENT
SPOKANE JUNKYARD AND ASSOCIATED PROPERTIES
SPOKANE, WASHINGTON

1. Introduction

This streamlined human health risk assessment (HHRA) evaluates risks associated with potential contact with residual polychlorinated biphenyls (PCBs) and lead contamination remaining in site soils following remediation. Cancer risks and potential adverse noncancer health effects associated with incidental ingestion of and dermal contact with PCBs were assessed using residential exposure assumptions. Exposures to lead were assessed by estimation of potential blood lead levels in children exposed to site soils. This streamlined HHRA was conducted in accordance with national and regional EPA guidance (EPA 1991, 1989).

2. Exposure Assessment

This streamlined HHRA assumed a residential exposure scenario based on EPA standard default reasonable maximum exposure (RME) residential factors (EPA 1991). Exposure factors and equations used to calculate contaminant intake for incidental ingestion and dermal contact are presented in Tables 1 and 2, respectively.

Risks were calculated for two scenarios: a "hot spot" scenario using the maximum detected concentration of each contaminant; and a scenario using an average concentration of each contaminant, calculated as the 95% upper confidence limit (UCL) on the arithmetic mean. For the purpose of this streamlined HHRA, the detection limit was used as a surrogate concentration in samples where lead or PCBs were not detected. A lognormal distribution was assumed when calculating the 95% UCL (EPA 1992c). All data points reported in the Spokane Junkyard Final Confirmation Sample Data spreadsheet provided by EPA were used in this evaluation.

3. Toxicity Assessment

PCBs. Toxicity values for PCBs were obtained from the Integrated Risk Information System database

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3. Toxicity Assessment

PCBs. Toxicity values for PCBs were obtained from the Integrated Risk Information System database (IRIS, EPA 1997) and from the PCB Cancer Dose-Response Assessment document (EPA 1996). This

guidance document describes the most recent approach for assessing carcinogenic risks associated with PCBs.

An oral reference dose (RfD) for Aroclor 1254 was available from IRIS (EPA 1997). This value was used to evaluate potential adverse noncancer health effects associated with incidental ingestion of PCBs. Reference doses are only available for Aroclor 1016 and Aroclor 1254; the value for Aroclor 1254 is lower (i.e., more conservative) and was therefore selected as a surrogate to represent the entire range of PCBs in this streamlined HHRA.

Because EPA has not promulgated dermal route toxicity values, oral route RfDs and SFs may be used to evaluate exposures to substances by the dermal route. Such route-to-route extrapolation has a scientific basis because once a chemical is absorbed, its distribution, metabolism, and elimination patterns (biokinetics) are usually similar, regardless of the exposure route. However, dermal toxicity values typically are based on absorbed dose, whereas oral exposures usually are expressed in terms of administered dose. Consequently, if adequate data concerning the gastrointestinal absorption of a chemical are available, then dermal RfDs and SFs may be derived by applying a gastrointestinal absorption factor to the oral toxicity value.

In this streamlined HHRA, the following methodology was employed for dermal exposures to soil. Dermal SFs were derived by dividing oral SFs by the fraction of the administered dose absorbed across the gastrointestinal tract, whereas dermal RfDs are derived by multiplying oral RfDs by the gastrointestinal absorption fraction. Dietary studies indicated that approximately 89% of the administered dose of Aroclor 1254 is absorbed across the gastrointestinal tract (Environmental Criteria and Assessment Office [ECAO] 1994); therefore, a gastrointestinal absorption fraction of 0.89 was applied to the oral RfD (2×10^{-5} mg/kg-day) to yield a dermal RfD of 1.8×10^{-5} mg/kg-day. The oral SF was not modified as described below.

For PCBs, recent guidance from EPA (1996) recommends using various slope factors depending upon the type of PCB mixture and the exposure route. For this risk assessment, the same slope factor was used for assessing risks associated with all exposure pathways considered. The upper-bound slope factor of 2 $(mg/kg/day)^{-1}$ was used to assess risks associated with soil ingestion and dermal contact. Because this guidance recommends this slope factor for each of these pathways, modifications based on available oral absorption information (e.g., for dermal contact) were not required.

Lead. A meaningful oral RfD cannot be developed for lead because many of this metal's noncancer effects may not exhibit a threshold in young children. Additionally, young children often are exposed to

lead concurrently via several environmental media. Recognizing this multimedia, multipathway potential for exposure, EPA developed the IEUBK Model to evaluate childhood risks associated with lead exposure (EPA 1994).

Young children represent the segment of the population at greatest risk from lead exposure. The reasons for children's relatively high sensitivity are twofold. First, compared to that of adults, children's intake of lead from the gastrointestinal tract is greater (50% for children versus 6% for adults). Second, children's developing organ systems are more sensitive to lead's toxic effects.

The IEUBK Model predicts the childhood blood lead levels expected to result from exposure to lead in soil and other media (i.e., air, water, diet, dust, and paint; EPA 1994). EPA recommends a benchmark of either 95% of the sensitive population of children having blood lead levels below 10 µg/dL or a 95% probability of an individual child having a blood lead level below 10 µg/dL. Using default parameters, the model predicts a screening concentration of approximately 400 mg/kg of lead in soil. In this streamlined HHRA the maximum and average site lead concentrations were used as soil concentrations in the IEUBK model to predict blood lead concentrations in children. The dust concentration was set to 70% of the soil concentration; all other parameters were left as the default values.

4. Risk Characterization

Potential excess lifetime cancer risks and noncancer hazard quotients associated with exposure to PCBs are presented in Table 3. Excess lifetime cancer risks are summed across pathways for each exposure scenario. Hazard quotients are also summed to calculate a hazard index (HI).

Federal environmental laws and regulations recognize that estimates of very small levels of risk are insignificant. The concept of *de minimis* risk refers to a specific level below which risks are so small that they are not of concern. In risk assessment, government agencies recognize that potential cancer risks less than 1×10^{-6} are generally *de minimis* and that risks between 1×10^{-6} and 1×10^{-4} are within the generally acceptable range. The point of departure from the range of acceptable risks generally is regarded as 1×10^{-4}. The EPA Superfund program has adopted these regulatory ranges, which are used to place the estimated potential excess lifetime cancer risks into context (EPA 1992b).

For evaluating noncarcinogenic effects, EPA defines acceptable exposure levels as those to which the human population, including sensitive subgroups, may be exposed without adverse effects during a lifetime or part of a lifetime, incorporating an adequate margin of safety. This acceptable exposure level is approximated best by an HI equal to 1. If the HI is less than 1, adverse effects usually would not be

expected. However, adverse effects may occur when the HI exceeds 1.

Excess lifetime cancer risks associated with exposure to PCBs totaled 8×10^{-6} and 3×10^{-6} using maximum and average soil concentrations, respectively, as shown in Table 3. These risks are within the range of cancer risks considered generally acceptable by EPA.

HIs associated with exposure to PCBs totaled 0.5 and 0.2 using maximum and average soil concentrations, respectively. These HIs are below the benchmark of 1; consequently, adverse noncancer health effects would be unlikely.

The IEUBK model was used to estimate potential blood lead concentrations in children exposed to site soil. In both scenarios, blood lead concentrations were below the benchmark of 10.0 µg/dL at the 95th percentile. Therefore, lead concentrations at the site should not pose an unacceptable risk. Histograms displaying the blood lead probability distributions using the maximum and average soil concentrations are presented in Figures 1 and 2, respectively.

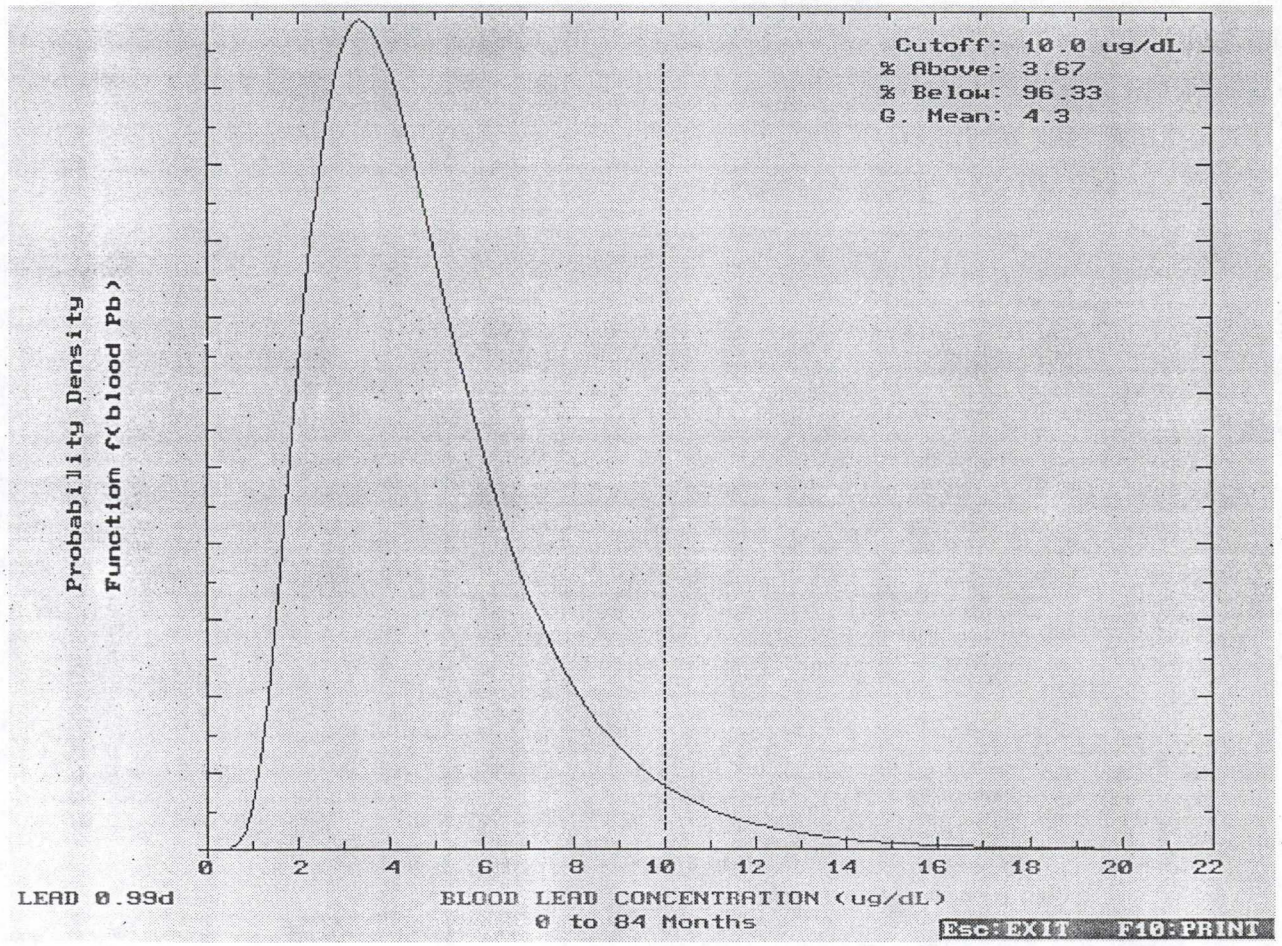

Figure 1 - Blood lead concentrations estimated using maximum soil concentration

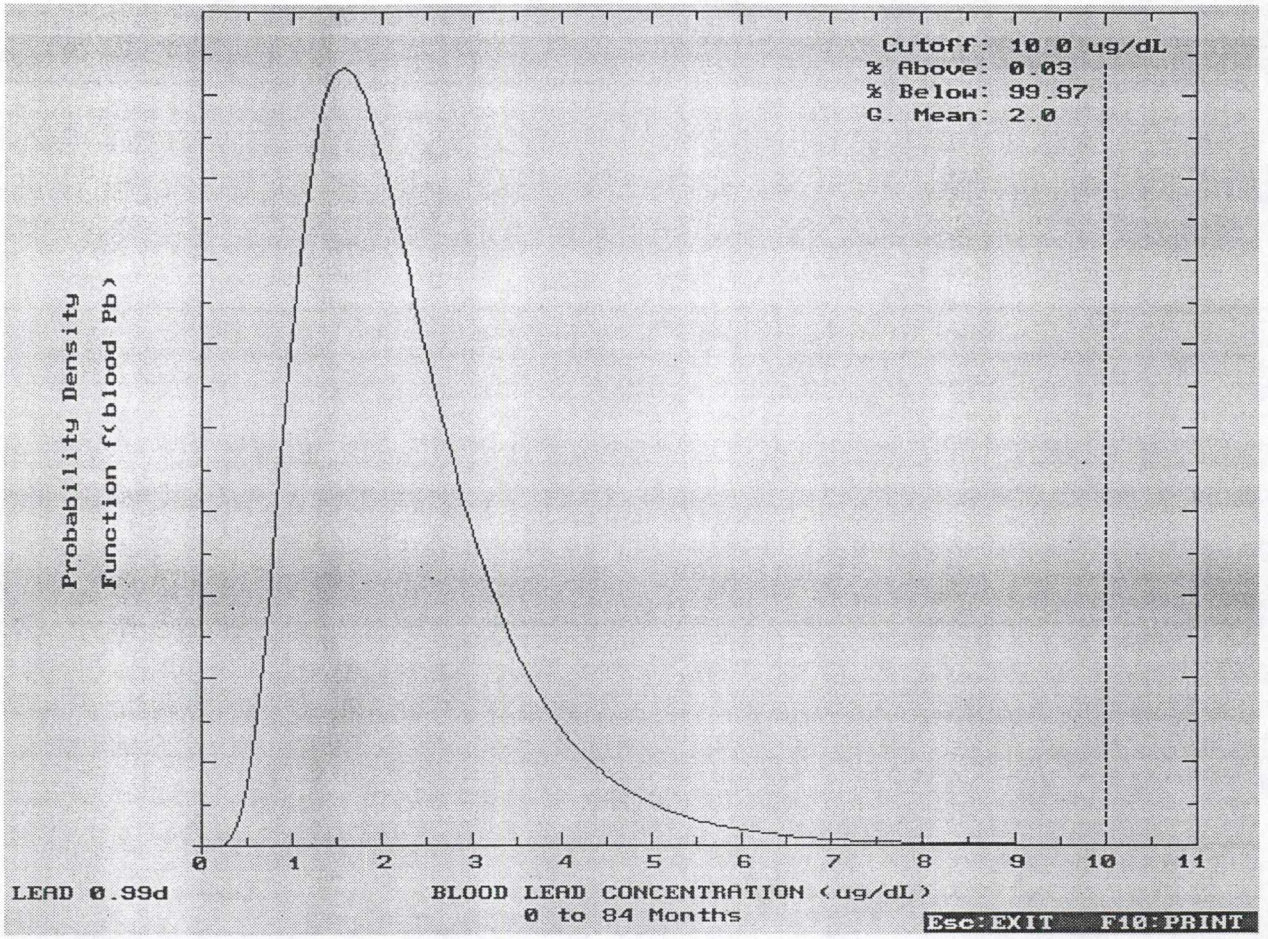

Figure 2 - Blood lead concentrations estimated using average soil concentration

5. References

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Table 1

INCIDENTAL INGESTION OF CONTAMINANTS IN SOIL
STREAMLINED HUMAN HEALTH RISK EVALUATION
SPOKANE JUNKYARD
SPOKANE, WASHINGTON

Equation:

$$SIF_{ing} = \frac{CF \times EF \times FI}{AT \times 365\ days/yr} \times \left[\frac{IR_{soil/child} \times ED_{child}}{BW_{child}} + \frac{IR_{soil/adult} \times ED_{adult}}{BW_{adult}} \right]$$

where:

SIF_{ing} = Summary intake factor for ingestion of contaminants in soil (day^{-1})
CF = Conversion factor (10^{-6} kg/mg)
EF = Exposure frequency (days/yr)
$IR_{soil/child}$ = Child soil ingestion rate (mg/day)
FI = Fraction of soil ingested from the contaminated source
ED_{child} = Exposure duration for a child (years)
$IR_{soil/adult}$ = Adult soil ingestion rate (mg/day)
ED_{adult} = Exposure duration for an adult (years)
AT = Averaging time (yrs)
BW_{child} = Body weight for a child (kg)
BW_{adult} = Body weight for an adult (kg)

Variable	Value	Units	Rationale/Source
EF	350	days/year	Assumes two weeks vacation per year (EPA 1991).
$IR_{soil/child}$	200	mg/day	Childhood soil ingestion rate (1- to 6-year-old age group).
FI	1	unitless	Assumes all soil ingested per day is from the contaminated source.
ED_{child}	6	years	Entire duration of age group.
$IR_{soil/adult}$	100	mg/day	Adult soil ingestion rate (EPA 1991).
ED_{adult}	24	years	Reflects 90th percentile duration at a single residence (30 years) less 6 years for child exposure duration (EPA 1991).
AT	70/30	years	Reflects a 70-year lifetime for carcinogenic effects and equal to exposure duration for noncarcinogenic effects (EPA 1991).
BW_{child}	15	kg	Average for 1- to 6-year old age group (EPA 1991).
BW_{adult}	70	kg	Average adult body weight (EPA 1991).

Carcinogenic SIF_{ing} (day^{-1}) = 1.6×10^{-6}
Noncarcinogenic SIF_{ing} (day^{-1}) = 3.7×10^{-6}

Note: Intake (mg/kg-day) = Concentration in soil (mg/kg) × SIF_{ing} (day^{-1}).

Table 2

DERMAL CONTACT WITH CONTAMINANTS IN SOIL
STREAMLINED HUMAN HEALTH RISK EVALUATION
SPOKANE JUNKYARD
SPOKANE, WASHINGTON

Equation:

$$SIF_d = \frac{CF \times EF \times AF \times ABS \times FC \times}{AT \times 365\ days/yr} \left[\frac{SA_{soil/child} \times ED_{child}}{BW_{child}} + \frac{SA_{soil/adult} \times ED_{adult}}{BW_{adult}} \right]$$

where:

SIF_d = Summary intake factor for dermal contact with contaminants in soil (day^{-1})
CF = Conversion factor (10^{-6} kg/mg)
EF = Exposure frequency (events/yr)
AF = Soil to skin adherence factor (mg/cm^2)
ABS = Absorption factor (unitless)
$SA_{soil/child}$ = Child skin surface area available for contact (cm^2/event)
FC = Fraction of soil contacted from the contaminated source
ED_{child} = Exposure duration for a child (years)
$SA_{soil/adult}$ = Adult skin surface area available for contact (cm^2/event)
ED_{adult} = Exposure duration for an adult (years)
AT = Averaging time (yrs)
BW_{child} = Body weight for a child (kg)
BW_{adult} = Body weight for an adult (kg)

Variable	Value	Units	Rationale/Source
EF	350	days/year	Assumes two weeks vacation per year (EPA 1991).
AF	1.0	mg/cm^2	EPA 1991
ABS	0.06	unitless	Absorption factor for PCBs (EPA 1992a).
$SA_{soil/child}$	3,900	cm^2	Area of hands, arms, legs, and feet of a child (EPA 1992a).
FC	1	unitless	Assumes all soil contacted per day is from the contaminated source.
ED_{child}	6	years	Entire duration of age group (EPA 1991).
$SA_{soil/adult}$	3,200	cm^2	Area of head, hands, and forearms of an adult (EPA 1992a).
ED_{adult}	24	years	Reflects 90th percentile duration at a single residence (30 years) less 6 years for child exposure duration (EPA 1991).
AT	70/30	years	Reflects a 70-year lifetime for carcinogenic effects and equal to exposure duration for noncarcinogenic effects (EPA 1991).
BW_{child}	15	kg	Average for 1- to 6-year old age group (EPA 1991).
BW_{adult}	70	kg	Average adult body weight (EPA 1991).

Carcinogenic SIF_d (day^{-1}) = 2.2×10^{-6}
Noncarcinogenic SIF_d (day^{-1}) = 5.1×10^{-6}

Note: Intake (mg/kg-day) = Concentration in soil (mg/kg) × SIF_d (day^{-1}).

Table 3

PCB RISK CALCULATIONS
STREAMLINED HUMAN HEALTH RISK EVALUATION
SPOKANE JUNKYARD
SPOKANE, WASHINGTON

Exposure Route	Exposure Point Concentration (mg/kg)	Carcinogenic Summary Intake Factor (day^{-1})	Slope Factor $(mg/kg\text{-}day)^{-1}$	Cancer Risk	Noncarcinogenic Summary Intake Factor (day^{-1})	Reference Dose (mg/kg-day)	Hazard Quotient
Maximum Detected Concentration Scenario							
Ingestion	1.11	1.6E-06	2	3.5E-06	3.7E-06	0.00002	0.20
Dermal	1.11	2.2E-06	2	4.8E-06	5.1E-06	0.000018	0.31
Total				**8.3E-06**			**0.52**
Average Concentration Scenario							
Ingestion	0.36	1.6E-06	2	1.1E-06	3.7E-06	0.00002	0.07
Dermal	0.36	2.2E-06	2	1.6E-06	5.1E-06	0.000018	0.10
Total				**2.7E-06**			**0.17**